

Tumors of the liver, gallbladder, and biliary tract are among the most common tumors worldwide. Neoplasms of the hepatobiliary system are classified as primary tumors, such as hepatocellular carcinoma and cholangiocarcinoma, or secondary lesions that result from the metastatic spread of malignant cells of nonhepatobiliary origin. Actually, there is only limited ability to reliably detect such lesions at early stages. Therefore, the clinical outcome of all these malignancies remains poor because patients usually present with advanced, often unresectable neoplasms. Consequently, hepatobiliary cancers impose a major socioeconomic burden on modern societies. However, during the last decades our understanding of the pathogenetic events underlying formation of liver cancer and gallbladder outgrowth has improved considerably. In particular, novel insights in the functional role of molecular mediators driving hepatobiliary cancerogenesis and the advances in understanding of the contribution of different cell subpopulations in cancer biology rose incredibly. Based on this knowledge, numerous novel potential biomarkers were discovered that will help to decrease the gap between the time points from initiation and detection of cancer.

The present synopsis contains 33 short editorials, commentaries, and correspondences previously published in journals of the *AME Publishing Company*. These contributions discuss or highlight recent articles that significantly contributed to the progression of knowledge on the pathogenesis or diagnosis of hepatobiliary cancer. In particular, the focus of these contributions are research topics and clinical contributions investigating issues contributing to the aggressiveness, heterogeneity, and tumorigenicity during initiating and progression of hepatocellular carcinoma and development of gallbladder cancer (*Figure 1*). The individual contributions were written by outstanding key leaders in their field.

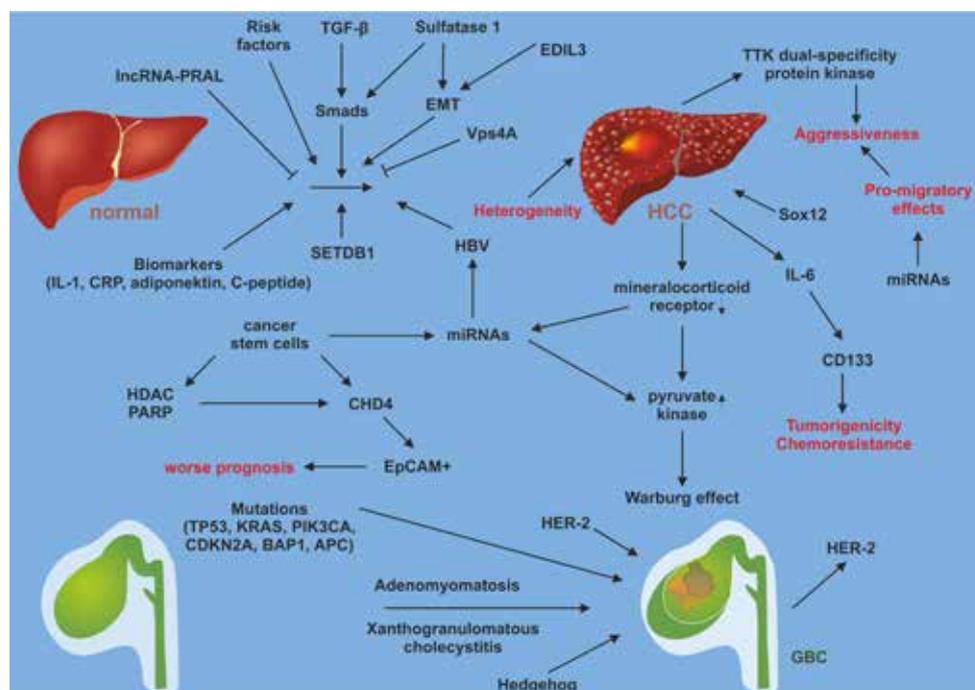


Figure 1 New perspectives in liver and gallbladder cancerogenesis. This book contains expert commentaries on mediators and signalling pathways contributing to the pathogenesis of hepatic and biliary tract neoplasms. The individual contributions discuss research highlights and provide short updates on new progress of specific fields that are of current interest in pathogenesis, diagnostic, and therapy of hepatobiliary cancer. Abbreviations used are: APC, adenomatous-polyposis-coli; BAP1, breast cancer 1 gene-associated protein-1; CD133, cluster of differentiation 133 (prominin-1); CDKN2A, cyclin-dependent kinase Inhibitor 2A; CHD4, chromodomain-helicase-DNA-binding protein 4; CRP, C-reactive protein; EDIL3, Epidermal growth factor-like repeats and discoidin domains 3; EMT, epithelial-mesenchymal transition; EpCAM, epithelial cell adhesion molecule; GBC, gallbladder cancer; HCC, hepatocellular carcinoma; HDAC, histone deacetylase; HER-2, human epidermal growth factor receptor 2; IL-1/6, interleukin-1/6; KRAS, Kirsten rat sarcoma viral oncogene; lncRNA-PRAL, long non-coding RNA-p53 regulation-associated lncRNA; miRNA, micro RNA; PARP, poly(ADP-ribose) polymerase; PIK3CA, phosphatidylinositol-4,5-bisphosphate 3-kinase catalytic subunit α ; Sox12, SRY-box 12; TGF- β , transforming growth factor- β ; TP53, tumor protein 53; TTK, dual-specificity protein kinase.

I think that this synopsis of short contributions will provide a good overview of current “hot topics” presently taking the attention in basic science and clinical practice. In addition, this compilation could serve as a possible starting point for those readers attending to increase their knowledge by further readings of up-to-date references cited in the individual contributions of this book.

I cordially thank the experts that contributed to this synopsis and the highly efficient editorial team of the *AME Publishing Company* helping in realizing this marvellous compilation. In particular, I am grateful to Elva S. Zheng for the extraordinary editorial support throughout the preparation of this textbook.

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